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Jee In Kim et al., J Food Nutr Disor 2017, 6:5
DOI: 10.4172/2324-9323-C1-006

11TH OBESITY AND ENDOCRINOLOGY CONGRESS

July 17-18, 2017 Melbourne, Australia

A pan-histone deacetylase inhibitor valproic acid prevented high fat diet-induced hypertension in mice

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Obesity is an epidemic increasing its number worldwide due to westernized high fat diet and one of the major risk factors of hypertension. Histone deacetylases (HDAC) involve in gene expression through regulating histone protein deacetylation. HDAC inhibitors are known to exert anti-cancer, anti-inflammatory effect and play protective role in cardiovascular diseases. In the present study, we applied high fat diet (HFD) mice model to mimic human obesity and observed increased body weight and higher blood pressure of HFD-fed mice compared with normal diet (ND)-fed mice. Then hypothesized that valproic acid (VPA), a FDA-approved HDAC inhibitor, might prevent HFD-induced hypertension. When HFD group reached to pre-hypertensive phase (130-140 mmHg systolic blood pressure), VPA was administered for 6 days (300 mg•kg-1/day). To evaluate the effect of VPA on developing HFD-induced hypertension, the expression of renin-angiotensin system (RAS) components and HDAC1 and acetylation of histone proteins were analyzed. VPA administration attenuated the progression of hypertension and altered RAS activation. In addition, increased HDAC1 in HFD-fed mice was reduced responding to VPA administration and lowered histone acetylation level was restored by VPA. The regulatory effect of HDAC1 on angiotensinogen expression was elucidated in MDCK cells using siRNA-mediated gene silencing system. In conclusion, VPA prevented HFD-induced hypertension through down-regulation of RAS activity following reduced HDAC1.

Biography

Jee In Kim has completed her PhD at the age of 37 years from Kyungpook National University and postdoctoral studies from Thomas Jefferson University School of Medicine. She is the Associate professor at Keimyung University School of Medicine. She has published more than 38 papers in reputed journals and has been serving as an secretary-general at Medical Research Center of Keimyung University School of Medicine.

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